

## I. AMENDMENTS

### IN THE CLAIMS

Cancel claims 3, 5, 6, 10, 13-15, and 19 without prejudice to renewal.

Please enter the amendments to claims 1, 4, 8, 11, 18, and 24, as shown below.

1. (Currently amended) A method for detecting an amyloid peptide-related neurological disorder in a ~~non-human animal~~ transgenic mouse model of the disorder, the method comprising:  
detecting a level of a calcium-responsive gene product in ~~brain~~ hippocampal tissue of the transgenic mouse animal model, wherein the calcium-responsive gene product is selected from a calbindin polypeptide, a neuropeptide Y polypeptide, an  $\alpha$ -actinin II polypeptide, a Fos polypeptide, an Arc polypeptide, a phospho-ERK polypeptide, a calbindin mRNA, a neuropeptide Y mRNA, an  $\alpha$ -actinin II mRNA, and a Fos mRNA, an Arc mRNA, and a phospho-ERK mRNA,  
and wherein the genome of said transgenic mouse comprises a transgene encoding a mutant amyloid precursor protein;  
wherein detection of a level of calcium-responsive gene product in the ~~brain~~ hippocampal tissue that differs from a level of the calcium-responsive gene product associated with a normal control mouse animal is indicative of an amyloid peptide-related neurological disorder in the mouse animal.

2. (Original) The method of claim 1, wherein the non-human animal model is an hAPP<sub>FAD</sub>/A $\beta$  transgenic non-human animal model of Alzheimer's Disease.

3. (Canceled)

4. (Currently amended) The method of claim 1 [[3]], wherein the brain tissue is dentate gyrus.

- 5.-6. (Canceled)

7. (Previously presented) The method of claim 1, wherein the neurological disorder is impaired spatial learning or impaired memory.

8. (Currently amended) A method for identifying a candidate agent for treating an amyloid peptide-related neurological disorder, the method comprising:

administering a test agent to a ~~non-human animal~~ transgenic mouse model of an amyloid peptide-related neurological disorder, wherein the genome of said transgenic mouse comprises a transgene encoding a mutant amyloid precursor protein; and

detecting a level of a calcium-responsive gene product *in vitro* in brain hippocampal tissue of the mouse animal, wherein the calcium-responsive gene product is selected from a calbindin polypeptide, a neuropeptide Y polypeptide, an  $\alpha$ -actinin II polypeptide, a Fos polypeptide, an Arc polypeptide, a phospho-ERK polypeptide, a calbindin mRNA, a neuropeptide Y mRNA, an  $\alpha$ -actinin II mRNA, and a Fos mRNA, an Arc mRNA, and a phospho-ERK mRNA;

wherein detection of a level of calcium-responsive gene product in the ~~brain~~ hippocampal tissue that differs significantly from a level of the calcium-responsive gene product in the absence of the agent indicates that the test agent is a candidate agent for treating an amyloid peptide-related neurological disorder.

9. (Original) The method of claim 8, wherein the non-human animal model is an hAPP<sub>FAD</sub>/A $\beta$  transgenic non-human animal model of Alzheimer's disease.

10. (Canceled)

11. (Currently amended) The method of claim 8 [[10]], wherein the brain tissue is dentate gyrus.

12. (Previously presented) The method of claim 8, wherein the neurological disorder is impaired spatial learning or impaired memory.

13.-15. (Canceled)

16. (Previously presented) The method of claim 1, wherein the amyloid peptide-related neurological disorder is a behavioral deficit.

17. (Previously presented) The method of claim 8, wherein the amyloid peptide-related neurological disorder is a behavioral deficit.

18. (Currently amended) A method for detecting an amyloid peptide-related neurological disorder in a ~~non-human animal~~ transgenic mouse model of the disorder, the method comprising:

detecting a level of a calcium-responsive gene product of the animal model, wherein the ~~animal model is a~~ transgenic mouse model has ~~having~~ a genome comprising a transgene encoding ~~[[an]]~~ a mutant amyloid precursor protein;

wherein detection of a level of calcium-responsive gene product in hippocampal tissue of the transgenic mouse that differs from a level of the calcium-responsive gene product associated with a normal control mouse is indicative of an amyloid peptide-related neurological disorder in the mouse.

19. (Canceled)

20. (Previously presented) The method of claim 18, wherein the calcium-responsive gene product is selected from calbindin mRNA, calbindin protein, c-fos mRNA, Fos protein, Arc mRNA, Arc protein, neuropeptide Y mRNA, neuropeptide Y protein, ERK mRNA, phospho-ERK protein,  $\alpha$ -actinin II mRNA, and  $\alpha$ -actinin II protein.

21. (Previously presented) The method of claim 18, wherein the amyloid peptide-related neurological disorder is a behavioral deficit.

22. (Previously presented) The method of claim 18, wherein the neurological disorder is impaired spatial learning or impaired memory.

23. (Previously presented) The method of claim 18, wherein the hippocampal tissue comprises dentate gyrus.

24. (Currently amended) A method for identifying a candidate agent for treating an amyloid peptide-related neurological disorder, the method comprising:

administering a test agent to a ~~non-human animal~~ transgenic mouse model of the amyloid peptide-related neurological disorder, wherein the ~~animal model is a~~ transgenic mouse model has ~~having~~ a genome comprising a mutant amyloid precursor protein; and

detecting a level of a calcium-responsive gene product in a hippocampal tissue of the transgenic mouse;

wherein detection of a level of calcium-responsive gene product in the hippocampal tissue that differs significantly from a level of the calcium-responsive gene product in the absence of the agent indicates that the test agent is a candidate agent for treating an amyloid peptide-related neurological disorder.

25. (Previously presented) The method of claim 24, wherein the calcium-responsive gene product is selected from calbindin mRNA, calbindin protein, c-fos mRNA, Fos protein, Arc mRNA, Arc protein, neuropeptide Y mRNA, neuropeptide Y protein, ERK mRNA, phospho-ERK protein,  $\alpha$ -actinin II mRNA, and  $\alpha$ -actinin II protein.

26. (Previously presented) The method of claim 24, wherein the amyloid peptide-related neurological disorder is a behavioral deficit.

27. (Previously presented) The method of claim 24, wherein the neurological disorder is impaired spatial learning or impaired memory.

28. (Previously presented) The method of claim 24, wherein the hippocampal tissue comprises dentate gyrus.